The Phase-Transition Theory of Evolution: A Lyapunov–Attractor Formalization of Emergent Biological Form

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We formulate a phase-transition theory of evolution in which biological form is modeled as a Lyapunov-stable attractor regime in a high-dimensional state space. The neo-Darwinian equation describes intra-phase frequency change, not the origination of biological phases. We define a total biological state X(t) over genomic, epigenetic, developmental, phenotypic, and niche coordinates, evolving under nonlinear dynamics $dX/dt = F(X; \mu, \theta) + \sigma\Xi(t)$. A biological form is identified with an attractor A_i and basin B_i. Emergence occurs when a control parameter μ drives the system to a critical value μc at which the dominant Jacobian eigenvalue crosses zero, old stability is lost, and a new attractor basin becomes available. The transition is detected by discontinuity in an order parameter Ω, Lyapunov instability of the prior regime, and stabilization of the successor regime. Natural selection is formally restricted to intra-basin frequency dynamics. The theory predicts critical slowing down, increased variance, increased autocorrelation, constraint-density thresholds, and spectral signatures of autocatalytic or developmental closure before major biological transitions.

Keywords: phase-transition evolution, emergence, Lyapunov stability, attractor states, biological form, macroevolution, nonlinear dynamics, criticality, self-organization, constraint closure, neo-Darwinian synthesis.

1. State Space

Let the total biological state be

$X(t) \in M$

where

$M = G \times E \times D \times P \times N$

G denotes genomic state, E epigenetic state, D developmental state, P phenotypic state, and N niche or ecological state.

Evolution is not restricted to allele-frequency change. It is motion in M.

The general system is

$dX/dt = F(X; \mu, \theta) + \sigma\Xi(t)$

F is endogenous biological dynamics. μ is a control parameter. θ denotes constraints. σΞ(t) denotes stochastic perturbation.

The Darwinian variable is a projection of X, not X itself.

Let πG be the genomic-frequency projection.

$\pi G: M \rightarrow \Delta n$

where Δn is an allele or variant-frequency simplex.

Neo-Darwinian dynamics are therefore lower-dimensional.

2. Biological Form

Definition 1. A biological form is a stable dynamical regime $A_i \subset M$ such that

$F(X; \mu, \theta) = 0$ for $X \in A_i$

and nearby trajectories converge to A_i.

The basin of A_i is

$B_i = \{X_o \in M : \lim t \rightarrow \infty X(t; X_o) \in A_i\}$

Thus form is not an aggregate of selected traits. Form is a stable regime of matter, information, development, and niche coupling.

Definition 2. A biological phase is the pair

$\Phi_i = (A_i, B_i)$

A phase is an attractor with its basin.

Definition 3. Macroevolutionary novelty is not a displacement within B_i. It is a transition

$\Phi_i \rightarrow \Phi_j$

with

$B_i \neq B_j$

and

$A_i \neq A_j$

3. Lyapunov Stability

For each phase Φ_i, let V_i be a Lyapunov function.

$V_i: B_i \rightarrow \mathbb{R} \geq 0$

with

$V_i(X) \geq 0$

$V_i(X) = 0$ iff $X \in A_i$

$dV_i/dt = \nabla V_i \cdot F(X; \mu, \theta) < 0$ for $X \notin A_i$

Hence an organismal regime persists because trajectories descend toward its attractor.

Persistence is not primary evidence of gradual assembly. Persistence is evidence of basin stability.

Proposition 1. If V_i exists with $dV_i/dt < 0$ on $B_i \setminus A_i$, then Φ_i is dynamically stable.

Proof. Standard Lyapunov convergence. The system cannot increase V_i along trajectories. The invariant set with $V_i = 0$ is A_i. Therefore trajectories in B_i approach A_i.

4. Emergence as Phase Transition

Let J be the Jacobian of F at A_i.

$J(A_i; \mu) = \partial F/\partial X \mid A_i$

Let λmax denote the eigenvalue of J with largest real part.

The phase Φ_i is locally stable when

$\text{Re } \lambda max[J(A_i; \mu)] < 0$

Criticality occurs when

$\text{Re } \lambda max[J(A_i; \mu c)] = 0$

After criticality, the old phase loses stability when

$\text{Re } \lambda max[J(A_i; \mu)] > 0$

for μ beyond μc.

Definition 4. Emergence occurs when a control parameter μ crosses μc and the system undergoes attractor replacement.

$\Phi_i \rightarrow \Phi_j$

where Φ_i is destabilized and Φ_j becomes stable.

Thus emergence is not pruning. Emergence is regime conversion.

5. Order Parameter

Let Ω be an order parameter.

$\Omega = \Psi(X)$

Ω measures integrated organization.

Examples include autocatalytic closure, developmental canalization, multicellular integration, neural coherence, or niche-system coupling.

A discontinuous biological transition satisfies

$\Delta\Omega c \neq 0$

where

$\Delta\Omega c = \lim \varepsilon \to 0^{+} \Omega(\mu c + \varepsilon) - \lim \varepsilon \to 0^{+} \Omega(\mu c - \varepsilon)$

If $\Delta\Omega c \neq 0$, novelty appears as a whole-state transition rather than incremental displacement.

Definition 5. A sudden biological emergence is a discontinuous change in Ω accompanied by attractor replacement.

6. Effective Potential

A minimal scalar model is

$d\Omega/dt = -\partial U(\Omega; \mu)/\partial \Omega$

Let

$U(\Omega; \mu) = a(\mu - \mu c)\Omega^{2} - b\Omega^{4} + c\Omega^{6}$

with

$a > 0$

$b > 0$

$c > 0$

Stable phases satisfy

$\Omega^{*}(\mu) = \text{argmin } U(\Omega; \mu)$

At $\mu = \mu c$, the global minimum may jump.

This models biological form as a stable minimum of an effective potential.

The organism is not assembled by summing advantages. It condenses into a new minimum.

7. Selection as Intra-Phase Dynamics

Within a stabilized phase, variant frequencies n_k obey

$dn_k/dt = n_k(f_k - \bar{f})$

where f_k is variant fitness and $\bar{f}$ is mean fitness.

This is the replicator equation.

It acts on frequencies within an already defined phase.

Selection maps

$B_i \rightarrow B_i$

Emergence maps

$B_i \rightarrow B_j$

Therefore selection is intra-phase. Emergence is inter-phase.

Proposition 2. Replicator dynamics alone do not define biological phase origination.

Proof. The replicator equation evolves n_k on a fixed simplex Δn. It presupposes the existence of variants, reproduction, inheritance, and fitness. These are already phase-level structures. Therefore the equation cannot explain the origin of the phase that makes the equation meaningful.

8. Whole-Before-Part Functionality

Let x_k be a component of X. Let r_k be its biological role.

$r_k = R_k(x_k, \Omega)$

Before the relevant phase transition, Ω is absent or below threshold.

$\Omega < \Omega c$

Therefore r_k may be undefined, unstable, or nonfunctional.

After transition,

$\Omega \geq \Omega c$

and

$r_k = R_k(x_k, \Omega)$

becomes stable.

Thus part-function is phase-relative.

A wing, membrane, eye, synapse, or genome segment is not fully defined outside the phase that gives it role.

Proposition 3. Biological function is not intrinsic to isolated parts.

Proof. Since r_k depends on Ω, and Ω is a phase-level variable, r_k is not determined by x_k alone. Function is assigned by the whole regime.

9. Genome as State-Space Constraint

Let developmental state z evolve by

$dz/dt = H(z; g, e, n)$

where g is genomic state, e epigenetic state, and n niche state.

Phenotype is

$p = Π(z)$

The genome does not directly specify p.

It constrains H.

Thus the genome is not a blueprint. It is a parameterization of the developmental vector field.

Let

$Hg = H(·; g, e, n)$

Different genomes alter the topology of developmental trajectories.

Genetic change modifies accessible basins. It does not by itself manufacture form.

10. Constraint Closure

Let KX be the active constraint matrix of the biological system.

Define constraint density

$C(X) = rank(KX)/dim(M)$

Emergence requires

$C(X) ≥ Cc$

where Cc is a critical closure threshold.

Below Cc, components are insufficiently coupled.

Above Cc, formerly independent degrees of freedom become mutually constrained.

This produces a new whole.

Definition 6. Constraint closure is the transition from weakly coupled component behavior to globally constrained regime behavior.

Constraint closure supplies an algebraic criterion for emergence.

11. Autocatalytic Transition

Let K be a catalytic interaction matrix.

Let δ denote degradation.

Net amplification is governed by

$K - \delta I$

Autocatalytic emergence occurs when

$\rho(K - \delta I) > 0$

where ρ is spectral radius.

Equivalently, the transition point is

$\lambda_{max}(K - \delta I) = 0$

Before this point, chemistry remains non-self-maintaining.

After this point, a self-amplifying regime exists.

This formalizes the transition from chemistry to metabolism.

12. Thermodynamic Compatibility

Biological order is local.

Let Ssys be system entropy and Senv environmental entropy.

Local organization requires

$dS_{sys}/dt < 0$

but thermodynamic admissibility requires

$dS_{total}/dt \geq 0$

where

$dS_{total}/dt = dS_{sys}/dt + dS_{env}/dt$

Therefore

$dS_{env}/dt > |dS_{sys}/dt|$

Biological emergence is compatible with the second law when internal order is coupled to entropy export.

Let I_{int} denote internal biological information or integrated constraint.

A dissipative biological phase satisfies

$dI_{int}/dt > 0$

and

$dS_{env}/dt > 0$

Life is not anti-entropic globally. It is locally ordering and globally dissipative.

13. Irreversibility

Let $\Delta U_{j\to i}$ be the barrier from phase Φ_j back to phase Φ_i.

The reversal probability is approximately

$P_{j\to i} \sim \exp(-\Delta U_{j\to i} / T_{eff})$

where T_{eff} is an effective fluctuation temperature.

As $\Delta U_{j\to i}$ increases,

$P_{j\to i} \to 0$

Thus evolutionary history is a ratchet of phase transitions.

Irreversibility is not merely historical contingency. It is barrier formation in state space.

14. Critical Signatures

Near transition,

$\mathrm{Re}\,\lambda_{max} \to 0$

Relaxation time diverges as

$\tau_{relax} \sim 1 / |\mathrm{Re}\,\lambda_{max}|$

This is critical slowing down.

Variance increases:

$\mathrm{Var}(\Omega) \uparrow$ as $\mu \to \mu c$

Autocorrelation increases:

$\mathrm{Corr}[\Omega(t), \Omega(t + \Delta t)] \uparrow$ as $\mu \to \mu c$

Susceptibility increases:

$\chi = \partial\Omega/\partial\mu$

$\chi \uparrow$ as $\mu \to \mu c$

Prediction 1. Major biological transitions should be preceded by increased fluctuation, increased variance, increased autocorrelation, and delayed recovery after perturbation.

Prediction 2. Experimental evolution near metabolic, developmental, or ecological thresholds should display abrupt attractor replacement rather than smooth accumulation.

Prediction 3. Fossil and developmental data should show long stability interrupted by short intervals of regime conversion.

Prediction 4. Genomic change should correlate with altered developmental landscape topology more strongly than with linear trait construction.

Prediction 5. Transition points should exhibit spectral signatures, especially eigenvalue crossing, increased network connectivity, and constraint closure.

15. Relation to Darwinian Dynamics

The Darwinian model is

variation → selection → accumulation → form

The phase-transition model is

state space → constraint saturation → Lyapunov instability → attractor transition → form

Darwinian theory tracks frequency.

Phase-transition theory tracks stability.

Darwinian formalism:

$dn_k/dt = n_k(f_k - \bar{f})$

Phase-transition formalism:

$dX/dt = F(X; \mu, \theta)$

$\text{Re}\ \lambda\max[J(A_i; \mu c)] = 0$

$\Delta\Omega c \neq 0$

$X(t) \rightarrow A_j$

Selection explains differential persistence within a phase.

It does not explain the origin of the phase.

16. Main Theorem

Theorem. If biological form is identified with Lyapunov-stable attractor phases in M, and if macroevolutionary novelty requires transition between distinct basins B_i and B_j, then natural selection is not the originative mechanism of biological form but an intra-phase frequency dynamic.

Proof. By Definition 1, form is a stable attractor A_i with basin B_i. By Definition 3, macroevolutionary novelty is $\Phi_i \rightarrow \Phi_j$ with $B_i \neq B_j$. Replicator selection maps variant frequencies within an already specified phase and therefore preserves the underlying phase space of the competing variants. Hence selection acts within B_i. Emergence requires transition to B_j. Therefore selection is not the originative mechanism of the new phase. It is an intra-phase dynamic.

17. Corollary

Corollary. The neo-Darwinian synthesis is mathematically adequate for microevolutionary optimization but ontologically insufficient for macroevolutionary origination.

Proof. Microevolution corresponds to motion within B_i. Macroevolutionary origination corresponds to $\Phi_i \rightarrow \Phi_j$. The former is frequency change. The latter is attractor replacement. These are not the same mathematical object.

18. Compressed Formal Statement

$X(t) \in M$

$M = G \times E \times D \times P \times N$

$dX/dt = F(X; \mu, \theta) + \sigma\Xi(t)$

$\Phi_i = (A_i, B_i)$

$V_i(X) \geq 0$

$dV_i/dt < 0$

$\text{Re}\ \lambda\max[J(A_i; \mu c)] = 0$

$\Delta\Omega c \neq 0$

$\Phi_i \rightarrow \Phi_j$

$dn_k/dt = n_k(f_k - \bar{f})$

selection: $B_i \rightarrow B_i$

emergence: $B_i \rightarrow B_j$

Therefore biological form is not pruned into existence. It appears as a phase transition into a new Lyapunov-stable attractor regime.

19. References

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